

LETTERS TO THE EDITOR

Right Ventricular Infarction Complicated by Right to Left Shunt

I

Manno et al. (1) described a case of right ventricular infarction complicated by right to left interatrial shunting, claiming that a similar case had not been described before. In fact, such a case was described by Morris and Donen in the *Archives of Internal Medicine* in September 1978 (Vol. 138, page 1405). The presence of right to left shunt across the atrium was documented by dye-dilution curve, and autopsy confirmed the presence of a patent foramen ovale.

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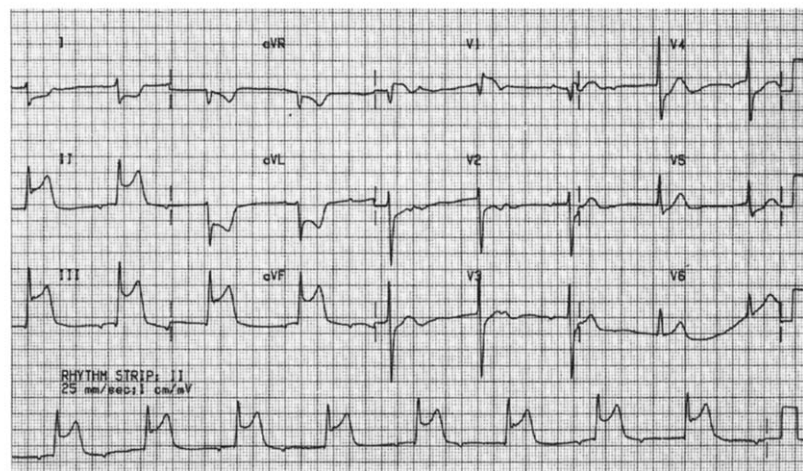
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II

The strong language of Phibbs' recent editorial on electrocardiographic reporting (1) encourages me to offer further comments on that lost art. A 12 lead tracing (Figure 1 in the article by Manno et al.) in the same issue bears the legend of "acute inferior infarction and high degree AV block." The rhythm shown is clearly an accelerated junctional one, with both antegrade and retrograde blocks and without opportunity for "AV" conduction.

Figure 1. From Manno et al. (2).



The same tracing is described in the text as "inferoposterior infarction," although the posterior component can only be imagined. Ironically, the pattern of ST segment elevation in lead V₁ and inferior infarction is, in fact, consistent with the authors' diagnosis of right ventricular infarction (2).

Finally, "serial electrocardiograms and creatine kinase isoenzyme determinations revealed evolution of transmural infarction." Here, at least, is something Phibbs did not know about transmural infarcts and their isoenzymes!

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- 2 Manno BV, Bemis CE, Carver J, Mintz GS. Right ventricular infarction complicated by right to left shunt. *J Am Coll Cardiol* 1983;1:554-7.
- 3 Chou T, Bel-Kahn J, Allen J, Brockmeier L, Fowler NO. Electrocardiographic diagnosis of right ventricular infarction. *Am J Med* 1981;70:1175-80.

III

To substantiate the presence of right ventricular infarction in their patient, Manno et al. referred to the findings of cardiogenic shock, heart block, hemodynamic evidence of a noncompliant right ventricle and echocardiographic and radionuclide angiographic signs of right ventricular dysfunction. We would like to call attention to the characteristic electrocardiographic changes of acute right ventricular infarction which are depicted in Figure 1 of their report. In addition to complete atrioventricular block and marked ST segment elevation in the inferior leads, there is also ST segment

elevation in lead V₁. ST segment elevation in one (lead V₁) or more of the conventional right precordial leads in the presence of acute left ventricular inferior myocardial infarction was found to be highly suggestive of coexisting right ventricular infarction (1). The recording of lead V₄R may further enhance the ability of the electrocardiogram to recognize this right ventricular lesion in patients who do not display the ST segment elevation in lead V₁ (2,3). Since this helpful sign is readily available from a routine test, it deserves full utilization.

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3. Croft CH, Nicod F, Corbett JR, et al. Detection of acute right ventricular infarction by right precordial electrocardiography. *Am J Cardiol* 1982;50:421-7.

Reply

We thank Shah for calling our attention to a previous report (1) of right to left shunt complicating inferior infarction.

Nikolic is correct in assessing the electrocardiographic rhythm as accelerated junctional, probably with 4:3 retrograde and 2:1 antegrade conduction; thus the term "high degree atrioventricular block" may be inappropriate in this setting. The tracing reveals in addition ST depression in anterior precordial leads (more pronounced in a subsequent tracing) accompanying inferior ST elevation. This has been reported to suggest posterior involvement (2). Thallium-201 myocardial scintigraphy at rest in this patient revealed posterior and inferior perfusion defects; thus we did not think it unreasonable to report this as inferoposterior infarction.

Chou, Fowler as well as Nikolic point out the importance of ST elevation in right precordial leads as evidence of right ventricular infarction. A recent report (3) has corroborated the high sensitivity, specificity and predictive accuracy of this finding.

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3. Klein HO, Tordjman T, Nimig R, et al. The early recognition of right ventricular infarction: diagnostic accuracy of the electrocardiographic V₄R lead. *Circulation* 1983;67:558-65.

Interaction of Calcium Channel and Beta-Adrenergic Blocking Agents

The combined administration of verapamil and propranolol or other beta-adrenergic blocking drugs is assuming increasing importance in treating severe angina. Increasing use is likely to bring problems due to drug interactions.

Winniford et al. (1) address this issue and note that the propranolol-verapamil combination was superior to propranolol alone. The point is made that the combination should be used cautiously because of potentially serious adverse effects, and the small incidence of such effects in their study was, at least in part, related to the exclusion from the study of patients with congestive heart failure or overt conducting system disease (1). This is entirely reasonable, as patients with these underlying problems are predictably prone to experience adverse effects of such a therapeutic combination.

However, it is important to note that adverse interactions between verapamil and beta-adrenergic blocking drugs may occur quite *unpredictably* in patients with relatively normal left ventricular function and absence of conducting system disease, who would not normally be expected to experience problems with such a combination. Our recent report (2) describes a group of patients with ischemic heart disease, no evidence of preexisting conducting system disease or heart failure and normal or only mildly impaired left ventricular function (assessed by subsequent cardiac catheterization or radionuclide angiography) who developed a shocklike syndrome with profound cardiac failure, hypotension and bradyarrhythmias soon after institution of combined oral therapy with verapamil and beta-adrenergic blocking drugs, and which resolved with cessation of the combined therapy. Since the report was submitted, we noted several additional patients under similar circumstances, with a variety of beta-adrenergic blocking drugs, including propranolol.

Such reactions were not clearly verapamil dose-dependent and no other factor apart from the addition of the second agent was found to account for the dramatic clinical deterioration in these patients and the subsequent improvement with termination of this therapy. Therefore, left ventricular function at rest, even when relatively preserved, and absence of overt conducting system disease, do not provide an accurate guide to tolerance of this drug combination.

Verapamil is an excellent antianginal agent with negative inotropic and chronotropic effects (3). Although its combination with a beta-adrenergic blocking drug may be contraindicated in certain obvious cases, this idiosyncratic, unpredictable adverse interaction as described above in otherwise "good candidates" for the combination is of some concern, and its incidence could well increase as the use of verapamil becomes more widespread. In this light, if the combination of a beta-adrenergic blocking drug and a calcium channel blocker is contemplated, then perhaps it may be preferable to use a less overtly negative inotropic and chronotropic calcium channel blocker, such as diltiazem or nifedipine, with which no such idiosyncratic effects have yet been noted.

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